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MYRIAD GENETICS INC.
INTELLECUTAL PROPERTY DEPARTMENT
320 WAKARA WAY
SALT LAKE CITY, UT 84108

EXAMINER	
LANDSMAN, ROBERT S	

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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Application Number: 10/035,344
Filing Date: January 04, 2002
Appellant(s): CIMBORA ET AL.

Jonathan Baker
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed 11/10/05 appealing from the Office action mailed 4/20/05.

(1) Real Party in Interest

A statement identifying by name the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

(3) Status of Claims

The statement of the status of claims contained in the brief is correct.

(4) Status of Amendments After Final

The appellant's statement of the status of amendments after final rejection contained in the brief is incorrect.

The amendment after final rejection filed on 7/20/05 has been entered.

(5) Summary of Claimed Subject Matter

The summary of claimed subject matter contained in the brief is correct.

(6) Grounds of Rejection to be Reviewed on Appeal

The appellant's statement of the grounds of rejection to be reviewed on appeal is correct.

(7) Claims Appendix

The copy of the appealed claims contained in the Appendix to the brief is correct.

(8) Evidence Relied Upon

No evidence is relied upon by the examiner in the rejection of the claims under appeal.

(9) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

(a) Claim Rejections - 35 USC § 112, first paragraph – enablement

Claims 1, 46 and 48-50 are rejected under 35 USC 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Appellants have asserted that AKT1 and AKT2 are involved in cell proliferation and apoptosis and that the claimed complexes can be used as therapeutic targets for such events. The claims recite both protein complexes as well as methods of using these complexes to screen for compounds which can modulate these interactions. However, Appellants have not taught how to use the present invention.

In In re Wands, 8USPQ2d, 1400 (CAFC 1988) page 1404, the factors to be considered in determining whether a disclosure would require undue experimentation include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims.

Page 8 of the specification states that:

> Aktl and Akt2 are serine/threonine protein kinases capable of phosphorylating a variety of known proteins. Aktl and Akt2 are activated by platelet-derived growth factor (PDGF), a growth factor involved in the decision between cellular proliferation and apoptosis. AKT kinases are also activated by insulin-like growth factor (1GF1), and in this capacity are involved in survival of cerebellar neurons). Furthermore, Aktl is involved in the activation of NFkB by tumor necrosis factor (TNF). Akt2 has been shown to be associated with pancreatic carcinomas. Akt kinases have been implicated in insulin-regulated glucose transport and the development of non-insulin dependent diabetes mellitus.

Appellants also provide Tables 1-10 in the specification, as well as numerous other paragraphs which discuss kinases (e.g. [0020] – [0030]), their interactions with other proteins as well as discussion of the vast array of potential roles of these kinases and various other proteins of the claimed complexes and methods. While Appellants have disclosed that the proteins of the invention are associated with numerous pathways and roles in a cell, they have not taught how the artisan is to use the complexes other than for the general purpose of screening for ligands which interfere with their formation. Appellants have not taught whether increases, or decreases, in specific complex formations would be beneficial, nor, regardless, what the results would allow the artisan to do with the knowledge. Much of Appellants disclosure uses phrases such as "involved in," "associated with," "likely plays a role in," as well as general functions such as "phosphorylating a variety of known proteins." This information does not provide sufficient guidance to teach the artisan how to use the present invention, especially in light of the fact that the claimed proteins do not have to be 100% identical to known proteins. However, reciting a function of these homologues would, itself, not remedy the situation.

Given the lack of guidance of what the specific functions are of the claimed complexes, as well as what useable information can be gathered from screening complexes to identify modulators of these interactions, it is unpredictable to the artisan how to use the information gathered from these screening methods. Claim limitations such as "promoting the interaction" and "inhibiting the interaction of said proteins" does not provide sufficient insight into how to use the claimed methods insofar as what useful conclusions can be gathered.

Furthermore, the breadth of the claims is excessive. The claims recite "a homologue at least 90% identical" to AKT1, AKT2, FNTA, TRPD, KIAA0728, PPL, Golgin-84, CL1C1, ARK7A2 and TPRD. Proteins which are "at least 90% identical" to the proteins of claim 1 would have one or more amino acid substitutions, deletions, insertions and/or additions to the full-length proteins. Appellants provide no guidance or working examples of proteins which are at least 90% identical to the full-length protein of the claims, nor do they provide a *function* of these proteins. Appellants have provided no guidance as to what critical residues are required to maintain the functional characteristics of AKT1, AKT2, FNTA, TRPD, KIAA0728, PPL, Golgin-84, CL1C1, ARK7A2 and TPRD. Furthermore, it is not predictable to one of ordinary skill in the art how to make a functional protein which is less than 100% identical to that of AKT1, AKT2, FNTA, TRPD, KIAA0728, PPL, Golgin-84, CL1C1, ARK7A2 and TPRD.

In summary, the breadth of the claims is excessive with regard to Appellants claiming "a homologue at least 90% identical" to AKT1, AKT2, FNTA, TRPD, KIAA0728, PPL, Golgin-84, CL1C1, ARK7A2 and TPRD. Applicants provide no guidance or working examples of proteins which are at least 90% identical to the full-length proteins of the claims, nor do they provide a *function* of these proteins. Furthermore, there is a lack of guidance of what the specific functions are of the claimed complexes, as well as what useable information can be gathered from screening complexes to identify modulators of these interactions. For these reasons, it is unpredictable to the artisan how to make functional complexes, or how to use the claimed screening methods, or the compounds identified by these methods. For these reasons, the Examiner holds that undue experimentation is required to practice the claimed invention.

(b) Claim Rejections - 35 USC § 112, first paragraph – written description

A. Claims 1, 46 and 48-50 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

These are genus claims. The claims recite "a homologue at least 90% identical" to AKT1, AKT2, FNTA, TRPD, KIAA0728, PPL, Golgin-84, CL1C1, ARK7A2 and TPRD (not TRPD). Proteins which are "at least 90% identical" to the proteins of claim 1 would have one or more amino acid substitutions, deletions, insertions and/or additions to the full-length proteins.

The specification and claims do not indicate what distinguishing attributes are shared by the members of the genus. Thus the scope of the claims includes numerous structural variants, and the genus

is highly variant because a significant number of structural differences between genus members is permitted. The specification and claims do not provide any guidance as to what changes should be made. Structural features that could distinguish compounds in the genus from others in the nucleic acid or protein class are missing from the disclosure. No common structural attributes identify the members of the genus. The general knowledge and level of skill in the art do not supplement the omitted description because specific, not general, guidance is what is needed. Since the disclosure fails to describe the common attributes or characteristics that identify members of the genus, and because the genus is highly variant, "AKT1, AKT2, FNTA, TRPD, KIAA0728, PPL, Golgin-84, CL1C1, ARK7A2 and TPRD" alone are insufficient to describe the genus. One of skill in the art would reasonable conclude that the disclosure fails to provide a representative number of species to describe the genus. Thus, Appellants was not in possession of the claimed genus at the time the invention was made.

(10) Response to Argument

(a) Claim Rejections - 35 USC § 112, first paragraph – enablement

Appellants argue that enablement is not precluded even if some experimentation is necessary, although the amount of experimentation needed must not be unduly extensive. Appellants further argue that the Examiner must provide a reasonable expectation as to why the claims are not enabled. Appellants cite references by Bascom, Qian, Aho, Valenzuela, Ventura, Wang, Ohira and Tsukahara in support of the fact that the structure and function of numerous proteins of the claimed Markush group are known.

These arguments have been considered, but are not deemed persuasive. While Bascom does disclose the structure and function of Golgi-84, there is no discussion of (1) what regions are critical to retain protein function. Though Qian does teach CLIC1, CLIC2 and CLIC3, again, there is only minimal information regarding the critical residues of these proteins (i.e. which residues are required to retain the function of the full-length proteins). Respectfully, the fact that the proteins are known to contain a nuclear localization signal is not persuasive since many proteins contain this signal. This signal alone, in the absence of other supporting information as to what other regions/residues of the protein must be maintained to retain protein function, is insufficient. The claims recite that up to 10% of the protein can be altered. Therefore, 90% of the protein must be retained. The nuclear localization signal constitutes only a small percentage of the overall protein and would not, itself, be expected to be sufficient to retain the activity of the full-length CLIC proteins.

Though the other reference do appear to teach a little more than both Bascom and Qian with regard to structure, there are no teachings in these reference, nor is there guidance or working examples in

the instant specification of what residues can be altered while retaining protein function, especially in light of the fact that the present claims do not provide and functional limitations. Appellants argue that Ventura teach that FTNA interacts with TBR-1 and ActR-1B and that ActR-1B has 90% homology to TbR1. However, Appellants have not provided any information regarding TbR1, so it is not clear, respectfully, how knowing the homology of TbR1 is related to the proteins claimed in the present invention. Finally, Appellants claim a protein called "KIAA0728" which is termed a "hypothetical protein" in the specification. No discussion regarding structure and function could be gathered from the specification, nor from the prior art regarding this hypothetical protein.

Appellants further argue that paragraph [0019] of the specification teaches the structure and function of AKT1 and cites numerous literature examples. Again, this argument has been considered, but is not deemed persuasive. While the function of AKT1 may very well be known, the question remains, as for the proteins discussed above, what residues can be altered while still retaining the functional capabilities of the wild-type protein. While the Table on page 23, as argued by Appellants, does provide some guidance and working examples of AKT1 proteins and other claimed proteins which are shorter than wild-type, it appears that these proteins are only functional insofar as they are used in a yeast two-hybrid assay as bait (AKT1) and prey (other claimed proteins). There is no demonstration that the AKT1 "fragment" used in this assay are functional. Again, the claims recite that AKT1 can be altered as much as 10% with no guidance or working examples in the specification as to what changes can be made. Furthermore, given this lack of guidance and working examples of AKT1 structure/function, as well as the minimal guidance as to the other proteins discussed above, it would not be predictable to the artisan what residues to alter and which to retain in order to maintain the functionality of the wild-type proteins. Furthermore, even if these residues were known, the specification still does not teach what the function is of the protein complex comprised of AKT1 and the proteins discussed above. The claims provide not functional limitations for the claimed protein complex. In addition, it would not be known by the artisan how to use this protein complex, nor is it taught in the specification whether the artisan would want to increase or decrease complex formation and for what purpose. Granted, the fact that two proteins are known to form a complex is interesting. However, in the absence of further guidance as to the significance of this interaction, the artisan, again, would not know how to use the complex.

Finally, Appellants argue that the fact that the Examiner withdrew the rejection under 35 USC 101 shows that the protein complexes have a use. However, the rejection under 35 USC 101 was only withdrawn in favor of a strengthened rejection under 35 USC 112, enablement. The rejection under 35 USC 101 was withdrawn since Appellants have provided a general utility for the protein complexes – that

is as therapeutic targets. While the rejection could have been maintained under 35 USC 101 as well, the Examiner determined that, since the functions of certain claimed proteins are were known at the time of the invention and that Appellants did, in a matter of speaking, provide a utility for the complexes, however, general, the Examiner decided that the rejection under 35 USC 112, first paragraph, enablement was the stronger rejection. Regardless of whether or not the utility of the complex is questionable, the issue of how to make and use the present invention is not.

It is believed that all pertinent arguments have been addressed.

(a) Claim Rejections - 35 USC § 112, first paragraph – written description

Appellants arguments are identical to those discussed above under 35 USC 112, first paragraph, enablement. These arguments have been considered, but are not deemed persuasive for the reasons discussed above.

(11) Related Proceeding(s) Appendix

No decision rendered by a court or the Board is identified by the examiner in the Related Appeals and Interferences section of this examiner's answer.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

Robert Landsman

ROBERT S. LANDSMAN, PH.D
PRIMARY EXAMINER

BRENDA BRUMBACK
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600

Conferees:

Brenda Brumback

Janet Andres

JANET L. ANDRES
SUPERVISORY PATENT EXAMINER